## Bambi DA

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#### **Hunting is key to deer population control**

University of Illinois 16 [Living with White-tailed Deer in Illinois, "Population Control," U of Illinois, http://web.extension.illinois.edu/deer/damage.cfm?SubCat=8890] AZ

To help limit deer damage, some form of deer population control is typically necessary. Well-regulated hunting through the state permit system, conducted in a safe manner, provides outdoor recreation to thousands of sportsmen and sportswomen in Illinois every year. Regulated hunting helps provide funding for other wildlife restoration efforts and outdoor recreation in Illinois, and is the primary method of achieving population management goals for Illinois’ white-tailed deer. Hunting The effective use of the legal hunting season is the best way to control deer populations. Harvesting deer during the regular archery and firearm deer hunting seasons may not solve problems completely, but it will be an important step toward long-term damage control. A very important goal of a hunting program on private land should be to harvest the maximum number of adult female deer (does). Killing male deer (bucks) accomplishes little to control the deer population. In addition to the reduction in deer densities, hunting can cause the dispersal of large, local concentrations of deer.

#### Handguns make hunting possible

Simpson 13 [Layne Simpson. “A Brief History of Handgun Hunting (with Whitetails in Mind).” FieldStream. http://www.fieldandstream.com/articles/hunting/2013/01/brief-history-handgun-hunting-whitetails-mind. No Date. January 2013. 12/15/15] KK

Originally designed as a defensive weapon for use against two-legged foes, the handgun did not begin to receive a great deal of attention from big-game hunters until the 1935 introductions of the .357 Magnum cartridge and a Smith & Wesson revolver chambered for it. Billed at the time as the world’s most powerful handgun, it was developed for law-enforcement use, and FBI director J. Edgar Hoover received one of the first to leave the factory. Then Major Douglas Wesson, who was a grandson of company co-founder Daniel B. Wesson, used a S&W revolver on highly publicized hunts for moose, elk, grizzly and antelope. The rest, as they say, is handgun-hunting history.

While Major Wesson’s adventures stirred up interest in hunting with a handgun, it really did not begin pick up a lot of momentum until about 20 years later. One of the biggest steps forward took place in 1955, when Smith & Wesson and Remington teamed up to produce the Model 29 revolver and the .44 Magnum cartridge. In addition to a muzzle energy rating close to twice that of the .357 Magnum, the fatter bullet punched a bigger hole through yon target. The .454 Casull, introduced two years later by Dick Casull and Jack Fulmer, upped muzzle energy to 2,000 ft.-lbs., making it the world’s most powerful handgun cartridge for about the next half century.

Up until the 1960s, the handgun even in its most powerful chambering was best suited for shots on game out to 100 yards or so, but that changed when handgun hunters began to experiment with rifle cartridges. One of the first was Alfred Goerg, a Washington state writer who bagged various and sundry game with a custom-built, single-shot pistol in .257 Roberts built on the Remington Rolling Block action. Possibly influenced by Goerg’s work with his single-shot pistol, Remington introduced its XP-100 in 1963. Its action was a modified version of the company’s Model 700 rifle action. Initially offered only in .221 Fireball, it eventually became available in more powerful chamberings such as the 7mm-08 and .35 Remington.

In 1967, Thompson/Center Arms began producing the Contender pistol. Initially chambered only for the .22 Long Rifle and .38 Special, its extremely sturdy tip-up action later proved to be capable of handling cartridges ranging in power from the .30-30 Winchester to the .45-70 Government. Hunters who took the time to practice enough to become proficient with the XP-100 and the Contender found it possible to cleanly take game at far greater distances than was practical with any revolver, especially when special handgun scopes with long eye relief eventually became available. They also proved to be more accurate--a top-quality single-shot pistol will shoot inside an inch at 100 yards.

While Remington’s XP-100 is no longer manufactured, plenty of other long-range handguns are available today, in a variety of great chamberings. I consider the .257 Roberts, 6.5 JDJ and .30-30 Winchester to be good to start with, while the 7mm-08 Remington, .308 Marlin Express, .309 JDJ and .308 Winchester are better choices for those who can handle the additional recoil. I consider a 14- to 15-inch barrel optimal, because anything shorter reduces velocity by an unacceptable amount, while a longer barrel can make the gun awkward to carry in the field.

#### **Deers are key hosts for disease spreading ticks.**

Stafford and Williams 14 [Kirby C. PhD. Chief Entomologist, State Entomologist. Scott C. PhD. Wildlife Biologist. “Deer, Ticks, and Lyme Disease: Deer Management as a Strategy for the Reduction of lyme Disease.” The Connecticut Agricultural Experiment Station. 2014. 12/10/15] KK

Ticks feed on blood and require an animal host to survive and reproduce. The blacklegged tick has four stages; egg, larva, nymph, and adult (male and female). This tick feeds on a wide variety of mammals and birds, although female ticks feed only on medium to large animal hosts, The larvae, nymphs, and adults feed only once and slowly; requiring 3-5 days to ingest the blood, depending on the stage of the tick. Larval I. scapularis are almost never infected with B. burgdorferi’. Larvae and nymphs typically become infected with Lyme disease bacteria when feeding on infected white-footed mice (Peromysms leucopus), chipmunks (T amias striatus), shrews (Sorax spp.), or certain species of birds [6-8]. The white-footed mouse is the principal source (reservoir) of B. burgdorferi, B. microti, and A. phagocytophilum [6, 8, 9]. While white- tailed deer are not reservoirs for Lyme disease and do not infect ticks with B. burgdozferi, these animals are the principal host for the adult ticks and overall tick abundance has been closely linked to the abundance of these animals [10-12]. Deer may have at least 10 to 50 female ticks attaching and dropping off each day through the fall and spring when adult ticks are active [13]. Each female tick lays around 2,000 eggs and then dies. While adult I. scapulan’s also will feed on other animal hosts ranging from dogs and cats to opossums (Didelphis virginiana), raccoons (Procyon Iotor), foxcs (Vulpes vulpes), coyotes (Cam‘s Iatrans), and skunks (Mephitis mephms), they do not feed on rodents and birds. These other larger animals each contribute only a small or modest fraction of the total engorged female ticks to the environment and 50-94% of all engorged female ticks are estimated to come from feeding on deer [14, 15]. It is questionable that I. scapularis can be maintained in significant numbers just from feeding on these medium-sized alternate animal hosts. Male Lwdas ticks do not require a blood meal and primarily seek female ticks on the animals to mate. Therefore, broadly speaking, deer are responsible for the reproductive success of the tick and mice and other reservoir hosts for the prevalence of infection with tick-home disease agents. However, larval and nymphal ticks also feed on deer and are important hosts for the immature stages as well. Deer are a dilution host as immature I. scapulan’s feeding on deer will not acquire B. burgdorferi. However, this is probably compensated by the number of ticks deer produce and disperse through the environment. White-tailed deer are the reservoir for Ehrlichia chafleensils, the causal agent for human monocyctotrophic ehrlichiosis or HME, which is transmitted by the lone star tick.

**Lyme disease is an epidemic that has no effective treatment. The impact is massive human suffering and huge monetary costs. And it is growing extremely fast.**

Mosher 15 [Dave. Deputy editor for Business Insider and Online director of Popular Science. He holds degrees in journalism and biology from The Ohio State University. “A ‘hidden epidemic’ in the US has ballooned into a public-health fiasco – and no solutions are in sight” Tech Insider. http://www.techinsider.io/lyme-disease-ticks-may-cost-billions-with-no-vaccine-2015-7. July 9, 2015. 1/14/16] KK

The US has an epidemic brewing within our borders, and the problem is much more serious than most people realize. Lyme disease is spreading fast, and it only takes the bite of a poppy-seed-size tick to contract. Even after treatment, symptoms can be difficult to shake. Those infected can develop severe, rheumatoid arthritis-like joint and muscle pain. Fatigue and neurological disorders — such as numbness, tingling, weakness, and cognitive impairment — can set in too. Left untreated, infections can lead to brain inflammation or heart problems. At least a handful of such cases have [proven fatal](http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6249a1.htm" \t "_blank). [A recent study](http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0116767) goes beyond human suffering inflicted by Lyme disease to estimate the monetary cost of this "hidden epidemic," as some [call it](http://www.ncbi.nlm.nih.gov/pubmed/18641487" \t "_blank). Researchers sifted through the health-insurance claims of 47 million people and discovered a staggering financial burden incurred by tens of thousands treated for Lyme disease — possibly more than $1 billion a year in the US alone. What's more, the mountain of data chips away at some longstanding mysteries surrounding Lyme disease: What kinds of symptoms people seek treatment for after a standard course of antibiotics, and how much diagnosis and treatment might predict these later symptoms. "Our study doesn't tell us anything about what better treatments are — but it tells us there's a big problem," says Dr. John Aucott, an author of the study and director of Johns Hopkins University's new [Lyme Disease Clinical Research Center](http://www.hopkinsrheumatology.org/specialty-clinics/lyme-disease-clinical-research-center/" \t "_blank). "We hope it changes the conversation. People, even five or 10 years ago, didn't think there was a problem."

#### Zoonotic diseases specifically lead to extinction

Casadevall 12 – Prof @ Department of Microbiology and Immunology and the Division of Infectious Diseases of the Albert Einstein College of Medicine Arturo. (“The future of biological warfare,” Microbial Biotechnology, p. 584-5)

In considering the importance of biological warfare as a subject for concern it is worthwhile to review the known existential threats. At this time this writer can identify at three major existential threats to humanity: (i) large-scale thermonuclear war followed by a nuclear winter, (ii) a planet killing asteroid impact and (iii) infectious disease. To this trio might be added climate change making the planet uninhabitable. Of the three existential threats the first is deduced from the inferred cataclysmic effects of nuclear war. For the second there is geological evidence for the association of asteroid impacts with massive extinction (Alvarez, 1987). As to an existential threat from microbes recent decades have provided unequivocal evidence for the ability of certain pathogens to cause the extinction of entire species. Although infectious disease has traditionally not been associated with extinction this view has changed by the finding that a single chytrid fungus was responsible for the extinction of numerous amphibian species (Daszak et al., 1999; Mendelson et al., 2006). Previously, the view that infectious diseases were not a cause of extinction was predicated on the notion that many pathogens required their hosts and that some proportion of the host population was naturally resistant. However, that calculation does not apply to microbes that are acquired directly from the environment and have no need for a host, such as the majority of fungal pathogens. For those types of host–microbe interactions it is possible for the pathogen to kill off every last member of a species without harm to itself, since it would return to its natural habitat upon killing its last host. Hence, from the viewpoint of existential threats environmental microbes could potentially pose a much greater threat to humanity than the known pathogenic microbes, which number somewhere near 1500 species (Cleaveland et al., 2001; Tayloret al., 2001), especially if some of these species acquired the capacity for pathogenicity as a consequence of natural evolution or bioengineering.

**Zoonotic diseases cause extinction – they circumvent barriers, become airborne, and don’t need living hosts**

**Quammen 12** David, award-winning science writer, long-time columnist for Outside magazine for fifteen years, with work in National Geographic, Harper's, Rolling Stone, the New York Times Book Review and other periodicals, 9/29, “Could the next big animal-to-human disease wipe us out?,” The Guardian, pg. 29, Lexis

**Infectious disease is all around us**. It's one of the basic processes that ecologists study, along with predation and competition. Predators are big beasts that eat their prey from outside. Pathogens (disease-causing agents, such as viruses) are small beasts that eat their prey from within. Although infectious disease can seem grisly and dreadful, **under ordinary conditions**, **it's** every bit as **natural** as what lions do to wildebeests and zebras. **But conditions aren't always ordinary**. Just as predators have their accustomed prey, so do pathogens. And just as a lion might occasionally depart from its normal behaviour - to kill a cow instead of a wildebeest, or a human instead of a zebra - so a pathogen can shift to a new target. **Aberrations occur**. When a pathogen leaps from an animal into a person, and succeeds in establishing itself as an infectious presence, sometimes causing illness or death, the result is a **zoonosis**. It's a mildly technical term, zoonosis, unfamiliar to most people, but it helps clarify the biological complexities behind the ominous headlines about swine flu, bird flu, Sars, emerging diseases in general, and the threat of a global pandemic. **It's** **a word** of the future, **destined for heavy use in the 21st century**. Ebola and Marburg are zoonoses. So is bubonic plague. So was the so-called Spanish influenza of 1918-1919, which had its source in a wild aquatic bird and emerged to kill as many as 50 million people. All of the human influenzas are zoonoses. As are monkeypox, bovine tuberculosis, Lyme disease, West Nile fever, rabies and a strange new affliction called Nipah encephalitis, which has killed pigs and pig farmers in Malaysia. Each of these zoonoses reflects the action of **a pathogen** that **can "spillover**", **crossing into people from other animals**. Aids is a disease of zoonotic origin caused by a virus that, having reached humans through a few accidental events in western and central Africa, now passes human-to-human. This form of interspecies leap is not rare; about 60% of all human infectious diseases currently known either cross routinely or have recently crossed between other animals and us. Some of those - notably rabies - are familiar, widespread and still horrendously lethal, killing humans by the thousands despite centuries of efforts at coping with their effects. Others are new and inexplicably sporadic, claiming a few victims or a few hundred, and then disappearing for years. Zoonotic pathogens can **hide**. The least conspicuous strategy is **to** lurk within what's called a reservoir host: a living organism that carries the pathogen while suffering little or no illness. When a disease seems to disappear between outbreaks, it's often still lingering nearby, within some reservoir host. A rodent? A bird? A butterfly? A bat? To reside undetected is probably easiest wherever biological diversity is high and the ecosystem is relatively undisturbed. The converse is also true: ecological disturbance causes diseases to emerge. Shake a tree and things fall out. Michelle Barnes is an energetic, late 40s-ish woman, an avid rock climber and cyclist. Her auburn hair, she told me cheerily, came from a bottle. It approximates the original colour, but the original is gone. In 2008, her hair started falling out; the rest went grey "pretty much overnight". This was among the lesser effects of a mystery illness that had nearly killed her during January that year, just after she'd returned from Uganda. Her story paralleled the one Jaap Taal had told me about Astrid, with several key differences - the main one being that Michelle Barnes was still alive. Michelle and her husband, Rick Taylor, had wanted to see mountain gorillas, too. Their guide had taken them through Maramagambo Forest and into Python Cave. They, too, had to clamber across those slippery boulders. As a rock climber, Barnes said, she tends to be very conscious of where she places her hands. No, she didn't touch any guano. No, she was not bumped by a bat. By late afternoon they were back, watching the sunset. It was Christmas evening 2007. They arrived home on New Year's Day. On 4 January, Barnes woke up feeling as if someone had driven a needle into her skull. She was achy all over, feverish. "And then, as the day went on, I started developing a rash across my stomach." The rash spread. "Over the next 48 hours, I just went down really fast." By the time Barnes turned up at a hospital in suburban Denver, she was dehydrated; her white blood count was imperceptible; her kidneys and liver had begun shutting down. An infectious disease specialist, Dr Norman K Fujita, arranged for her to be tested for a range of infections that might be contracted in Africa. All came back negative, including the test for Marburg. Gradually her body regained strength and her organs began to recover. After 12 days, she left hospital, still weak and anaemic, still undiagnosed. In March she saw Fujita on a follow-up visit and he had her serum tested again for Marburg. Again, negative. Three more months passed, and Barnes, now grey-haired, lacking her old energy, suffering abdominal pain, unable to focus, got an email from a journalist she and Taylor had met on the Uganda trip, who had just seen a news article. In the Netherlands, a woman had died of Marburg after a Ugandan holiday during which she had visited a cave full of bats. Barnes spent the next 24 hours Googling every article on the case she could find. Early the following Monday morning, she was back at Dr Fujita's door. He agreed to test her a third time for Marburg. This time a lab technician crosschecked the third sample, and then the first sample. The new results went to Fujita, who called Barnes: "You're now an honorary infectious disease doctor. You've self-diagnosed, and the Marburg test came back positive." The Marburg virus had reappeared in Uganda in 2007. It was a small outbreak, affecting four miners, one of whom died, working at a site called Kitaka Cave. But Joosten's death, and Barnes's diagnosis, implied a change in the potential scope of the situation. That local Ugandans were dying of Marburg was a severe concern - sufficient to bring a response team of scientists in haste. But if tourists, too, were involved, tripping in and out of some python-infested Marburg repository, unprotected, and then boarding their return flights to other continents, the place was not just a peril for Ugandan miners and their families. It was also an international threat. The first team of scientists had collected about 800 bats from Kitaka Cave for dissecting and sampling, and marked and released more than 1,000, using beaded collars coded with a number. That team, including scientist Brian Amman, had found live Marburg virus in five bats. Entering Python Cave after Joosten's death, another team of scientists, again including Amman, came across one of the beaded collars they had placed on captured bats three months earlier and 30 miles away. "It confirmed my suspicions that these bats are moving," Amman said - and moving not only through the forest but from one roosting site to another. Travel of individual bats between far-flung roosts implied circumstances whereby Marburg virus might ultimately be transmitted all across Africa, from one bat encampment to another. It voided the comforting assumption that this virus is strictly localised. And it highlighted the complementary question: why don't outbreaks of Marburg virus disease happen more often? Marburg is only one instance to which that question applies. Why not more Ebola? Why not more Sars? In the case of **Sars**, the scenario **could have been very much worse**. Apart from the 2003 outbreak and the aftershock cases in early 2004, it hasn't recurred. . . so far. Eight thousand cases are relatively few for such an explosive infection; 774 people died, not 7 million. Several factors contributed to limiting the scope and impact of the outbreak, of which humanity's good luck was only one. Another was the speed and excellence of the laboratory diagnostics - finding the virus and identifying it. Still another was the brisk efficiency with which cases were isolated, contacts were traced and quarantine measures were instituted, first in southern China, then in Hong Kong, Singapore, Hanoi and Toronto. **If the virus had arrived in a different** sort of big **city** - more loosely governed, full of poor people, lacking first-rate medical institutions - **it might have burned through a much larger segment of humanity**. One further factor, possibly the most crucial, was inherent in the way Sars affects the human body: symptoms tend to appear in a person before, rather than after, that person becomes highly infectious. That allowed many Sars cases to be recognised, hospitalised and placed in isolation before they hit their peak of infectivity. With influenza and many other diseases, the order is reversed. That probably helped account for the scale of worldwide misery and death during the **1918**-1919 **influenza**. And that infamous global pandemic **occurred** in the era **before globalisation**. Everything nowadays moves around the planet faster, including viruses. **When the Next Big One comes**, **it will** likely **conform to the** same perverse pattern as the **1918 influenza**: **high infectivity preceding notable symptoms**. That will help **it move through** cities and **airports like an angel of death**. The Next Big One is a subject that disease scientists around the world often address. The most recent big one is Aids, of which the eventual total bigness cannot even be predicted - about 30 million deaths, 34 million living people infected, and with no end in sight. Fortunately, **not every virus goes airborne** from one host to another. **If HIV**-1 **could**, **you and I might already be dead**. **If** the **rabies** virus **could**, it **would be the most horrific pathogen on the planet**. **The influenzas are well adapted for airborne** transmission, which is why a new strain can circle the world within days. The Sars virus travels this route, too, or anyway by the respiratory droplets of sneezes and coughs - hanging in the air of a hotel corridor, moving through the cabin of an aeroplane - and that capacity, combined with its case fatality rate of almost 10%, is what made it so scary in 2003 to the people who understood it best. Human-to-human **transmission is the crux**. **That** capacity **is what separates a** bizarre, awful, **localised**, intermittent and mysterious **disease** (such as Ebola) **from a global pandemic**. Have you noticed the persistent, low-level buzz about avian influenza, the strain known as H5N1, among disease experts over the past 15 years? That's because avian flu worries them deeply, though it hasn't caused many human fatalities. Swine flu comes and goes periodically in the human population (as it came and went during 2009), sometimes causing a bad pandemic and sometimes (as in 2009) not so bad as expected; but avian flu resides in a different category of menacing possibility. It worries the flu scientists because they know that H5N1 influenza is extremely virulent in people, with a high lethality. As yet, there have been a relatively low number of cases, and it is poorly transmissible, so far, from human to human. It'll kill you if you catch it, very likely, but you're unlikely to catch it except by butchering an infected chicken. But if H5N1 mutates or reassembles itself in just the right way, if it adapts for human-to-human transmission, it could become the biggest and fastest killer disease since 1918. It got to Egypt in 2006 and has been especially problematic for that country. As of August 2011, there were 151 confirmed cases, of which 52 were fatal. That represents more than a quarter of all the world's known human cases of bird flu since H5N1 emerged in 1997. But here's a critical fact: those unfortunate Egyptian patients all seem to have acquired the virus directly from birds. This indicates that the virus hasn't yet found an efficient way to pass from one person to another. Two aspects of the situation are dangerous, according to biologist Robert Webster. The first is that Egypt, given its recent political upheavals, may be unable to staunch an outbreak of transmissible avian flu, if one occurs. His second concern is shared by influenza researchers and public health officials around the globe: with all that mutating, with all that contact between people and their infected birds, the virus could hit upon a genetic configuration making it highly transmissible among people. "**As long as H5N1 is out there in the world**," Webster told me, "**there is the possibility of disaster**. . . There is the theoretical possibility that it can acquire the ability to transmit human-to-human." He paused. "And then God help us." We're unique in the history of mammals. **No other primate has ever weighed upon the planet to anything like the degree we do**. In ecological terms, we are almost paradoxical: large-bodied and long-lived but grotesquely abundant. **We are an outbreak**. **And here's the thing about outbreaks**: they end. In some cases they end after many years, in others they end rather soon. In some cases they end gradually, in others they end with a crash. In certain cases, they end and recur and end again. **Populations** of tent caterpillars, for example, seem to rise steeply and fall sharply on a cycle of anywhere from five to 11 years. The crash endings are dramatic, and for a long while they seemed mysterious. What could account for such sudden and recurrent collapses? One possible factor is infectious disease, and viruses in particular.